from Judea were allowed to go to Mecca on the annual pilgrimage, so it is somewhat of a mystery how the plague came to appear in Jeddah, but some hold to the opinion that as the annual travel was stopped by sea it must have been transmitted by the overland route by the way of the Persian Gulf from India.

I shall make reports from time to time of all the facts that I can gather about this disease, and should it reach this place, will cable for instructions as to what shall be done about goods shipped to the United States.

I have the honor to be, sir, your obedient servant,

W. W. MASTERSON,
United States Consul.

Hon. ASSISTANT SECRETARY OF STATE.

PLAGUE.

[Translated in this Bureau from the "Veröffentlichungen des Kaiserlichen Gesundheitsamtes," Berlin, July 7, 1897.]

ARABIA—*Djeddah.*—On June 10, 6 deaths from plague were reported in different quarters of the city, especially in Hadramanti. From June 11 to 16 there were on the several days 3, 1, 4, 3, 3, 3 deaths from plague.

BRITISH EAST INDIES—*Bombay.*—According to the Bombay Government Gazette there were in the four weeks, from May 19 to June 15, 56, 34, 34, and 26 deaths, respectively.

Macao.—According to advices from Hongkong, plague has not yet assumed an epidemic character in Macao. Only isolated cases appear in the vicinity of Canton.

JAPAN.—From April 13 to May 5 there were in the prefecture of Taipefu, in Kilung Sinchu, and some small places in north Formosa, 10 cases and 14 deaths, which occurred among Chinese, besides 2 suspicious cases and 1 suspicious death reported among Chinese and Japanese. In middle Formosa 1 case, 1 death occurred among the Chinese population; in south Formosa 6 cases, 4 deaths among Japanese and 163 cases, 109 deaths among Chinese. Total for the Island of Formosa, including suspicious cases, 184 cases and 130 deaths.

BRAZIL.

Sanarelli on the Germ of Yellow Fever.

[Continued.]

[Translated in this Bureau from the Portuguese.]

DECISIVE EXPERIMENTATION—VEHICLES AND CONTAGION.

Experiment in the human living subject must naturally encounter opposition and prejudice, but it would seem to be essential to the solving of problems of incalculable importance to the human race.

The autoexperiments of Pettenkofer, Emerich, Metchnikoff, and others, who voluntarily swallowed cultures of choleraic microbes, pointed out the way to the experimenter who would definitely settle a question of pathologic importance. Metchnikoff succeeded in inducing a typical attack of cholera in man by living cultures of Koch's vibrio, thus probably demonstrating the specific nature of the microbe after ten years of discussion. There have also been courageous experimenters in yellow fever. Repeated experiment in self-inoculation has been made directly with the products of the disease, but without in any case producing an attack of the disease. The cause of this ill success is now explained. Those who have made the attempt, believed the yellow fever virus

to be in the black vomit, and it was chiefly with this substance that their experiments were made. It is now evident that the icteroid bacillus finds its way into the stomach by means of hemorrhage, and that it exists in a state of dilution in the blood. My experiments were performed on five men.

For reasons that will be readily understood, living cultures were not used, but simply cultures in broth fifteen to twenty days old, filtered by a Chamberlain filter and sterilized with a few drops of formic aldehyde. In two individuals the injection was subcutaneous, and in three, intravenous. The results of these slight but perfectly successful experiments cast an unexpected light on the pathogenic mechanism of icteroid typhus, formerly so little understood. They are in brief, as follows:

Injection of filtered culture, in relatively small doses, reproduces in man typical yellow fever symptoms—fever, congestions, hemorrhages, vomit, steatosis of the liver, cephalalgia, rachialgia, nephritis, anuria, uræmia, jaundice, delirium, collapse—in short all the symptomatic and anatomic elements which constitute the basis for the diagnosis of yellow fever. This fact represents not only a brilliantly convincing argument in favor of the specific value of the icteroid bacillus, but a success of the first order in the rich field of experimental science.

The accepted theory represents the digestive system, especially the stomach, as the seat of the disease, simply because the gastrointestinal phenomena are strongly pronounced. Eliminate this theory, however, and demonstrate that these phenomena are due to a specific virus manufactured by a microbe and circulating in the blood, and yellow fever falls into its proper place among diseases of microbic origin.

All the symptomatic phenomena, all the functional alterations, all the anatomical lesions of yellow fever are the consequence of the steatogenic, emetic, and hemolytic substances fabricated by the icteroid bacillus.

The general symptoms of yellow fever, its ataxo-adynamic manifestations, its tendency to hæmorrhage, jaundice, etc., have caused it to be compared to poisoning by the venom of certain serpents.

Another point of contact between the two morbid processes is the hœmatogenous gastroenteritis, which in cases of serpent poisoning has been erroneously attributed to an effort to eliminate the poison from the system.

Having got rid of the purely arbitrary theory with regard to the mode of ingress and the proper seat of energy of the yellow fever virus, we have the difficult task of demonstrating the avenues of entrance of the microbe.

No literature exists in yellow fever countries sufficiently demonstrative in character to prove transmission by water. There is, however, an inexhaustible supply of facts demonstrating its atmospheric transmission.

The one example cited relative to the decrease of yellow fever in Vera Cruz after the installation of a good water supply system has only a relative value. The tendency is to attribute improvement in public health to one hygienic measure when it should rather be ascribed to a conjunction of sanitary improvements. The remarkable resistance of the icteroid bacillus to desiccation and to water, authorizes the admission of the diffusion of the yellow fever virus by air and water.

Contagion by the respiratory system has been proved by animal experiment.

With regard to the mechanism of contagion by water, it is an indubitable fact that the epithelia of the digestive system, when intact, do not in general permit the passage of any species of pathogenic germ. But it must be remembered that in our yellow fever countries the slightest disturbances of the digestive functions, such as the abuse of alcoholic drinks, ices, fruits, etc., especially on the part of recent arrivals, depress the system and prepare for the entrance upon the scene of yellow fever. It can not be denied that persons recently arrived in yellow fever countries are subject to catarrh of the biliary system, which, united with the over exertion of the liver, which accompanies it, may facilitate the entrance of the bacillus into the intestines. Nothing is easier than such an entrance of the bacillus at the season when it forms part of the microbic flora of the yellow fever locality.

We have now to study the origin, development, duration, and termination of those secondary infections which have so long and so obstinately contributed to obscure the true cause of yellow fever.

The icteroid bacillus, whether through effect of its specific virus or through the grave hepatic lesions which are its most immediate consequence, favors at a given moment the entrance into the organism of septic microbes, which not only terminate the disease much sooner than the specific microbe would do, but attack this specific agent, invading its domain, suppressing its vegetative faculty and even affecting its vitality. Consequently, this microbic antagonism between the yellow fever bacillus and septic infection, instead of being useful to the patient who represents the theatre of action, hastens his end.

TRANSMISSION OF YELLOW FEVER BY WAY OF THE SEA.

Explicative theory.—There is a curious biologic phenomenon which acquires an immense value in the epidemiology of yellow fever. The maritime propagation of yellow fever is now a fact so firmly established that we should direct our attention to discovering its cause, basing our researches on our knowledge of the specific microbe.

The behavior of the yellow fever microbe on board vessels differs greatly from that of the cholera vibrio under the same circumstances. When cholera is introduced on board a vessel it breaks out with a violence varying with the quantity and energy of the choleraic vibrio and with individual predisposition; after the outbreak the cholera vibrio does not seem to find in the ordinary conditions of maritime transportation a medium favorable to its existence. It rapidly yields to measures of disinfection and is extinguished. Yellow fever, on the contrary, once installed on board a vessel, maintains itself long and tenaciously, especially in summer, and is transferred to warehouses and stores. Old and much-used vessels are extremely unfit to be used in countries where yellow fever is endemic.

Those who are especially concerned with naval hygiene consider close, unventilated ships, in which the air is vitiated and the water in the hold fetid, to be veritable yellow fever factories.

Humidity, heat, and want of light appear, therefore, to be the agents best adapted to the preservation of the icteroid bacillus. But in the present state of our knowledge it is not possible to attribute to these agents alone a specific value, when other conditions exist which militate in its favor. We must, therefore, look for some other cause favoring in some way the nautical habitat of the icteroid bacillus.

A singular phenomenon, which frequently attracted my attention during these studies, suggested to me that the probable cause of this mysterious resistance of the icteroid bacillus is, that the fungus of common mould, in the atmosphere, protects the bacillus.

The microbe of yellow fever, although endowed with remarkable resistance in the presence of physico-chemical agents, can not remain indifferent to substances adopted to its nourishment.

It is certain that during its saprophytic existence outside of the organism, as during summer on board a vessel, it could not make use of nutritive principles of much value; it is also true that it has been sometimes found incapable of developing in ordinary gelatine. But if in close proximity to it there should develop an accumulation of vegetable mould, this would suffice for the nourishment and development of the bacillus which without it would be condemned to a more or less speedy death. This quality in mould of favoring the growth of the bacillus may be demonstrated experimentally by depositing the spores of such fungi on gelatine previously planted with the icteroid microbe, but which has remained for some time sterile. After the mould has commenced to develop its mycelium there soon appears a circle of small point-like colonies of the icteroid microbe. As the mycelium grows these colonies become more numerous, extending rapidly around the central nucleus of mycelium. After some days the gelatine in which the mould developed presents a singular appearance. Around each fungus the colonies of the bacillus, which might be supposed to be dead or incapable of further development, form a sort of constellation of colonies the more numerous the nearer they approach the point of first development. It would seem from this that there exists a radiating influence within which the microbe developes. Outside of this circle the microbic development ceases abruptly, and the remaining gelatine continues sterile. It is probable that this property is possessed by the fungi in general. It is also probable that there exist in nature, especially in localities in which yellow fever is installed in great vigor, fungi not now classified, which are endowed with a strong and specific potency. This strange phenomenon of parasitism, which may be called "a loan of the means of existence;" this curious form of soprophytism may represent the principal cause of the ready acclimatization of yellow fever on shipboard. Humid heat and lack of air are precisely the conditions most favorable to the growth of fungus, and are therefore indirectly favorable to the vitality of the icteroid bacillus. This property of symbiosis, which was observed by Metchnikoff, with regard to the cholera vibrio, is in accord with many observations made during the progress of a yellow-fever epidemic. We may therefore consider the fungi as the natural protectors of the specific agent of yellow fever, since it is only by their assistance that the microbe continues to live and multiply. The intervention of this factor, apparently so insignificant, may constitute the principal cause of the acclimatization of yellow fever, not only on board ship, but also in localities in which the conditions would not appear to be favorable to its development.

We are already aware that one of the factors considered indispensable to the development of yellow fever is humidity, which, accompanied by heat, forms the condition

required for the formation of fungus. It is in fact to defective ventilation and high hygrometric conditions of the atmosphere that the unhealthiness of Rio de Janeiro is attributed.

During the great epidemic of yellow fever of 1872 at Montevideo the disease showed an inexplicable predilection for attacking houses fronting to the north. Now the houses on the north side of streets in Montevideo are conspicuous for their dampness. It is, therefore, probable that the factor of humidity whether on board ships, along the coast or in the interior, is the principal coefficient in this biologic phenomenon, and not some commonplace meteorological influence. On the other hand the natural resistance of the icteroid bacillus to desiccation, nature's method of disinfection, and its longevity in sea water are sufficient to explain the acclimatization of icteroid typhus on ships and in maritime localities.

Extract from a paper entitled "Experimental and Anatomical Researches in Yellow Fever."

[Transmitted by Passed Assistant Surgeon H. D. GEDDINGS—Translated in this Bureau from the Annals de l'Institut Pasteur, Paris, June 25, 1897.]

By Dr. W. HAVELBURG, Rio de Janeiro.

The idea of looking for the specific germ of yellow fever in the contents of the stomach and intestines naturally suggests itself. Yellow fever begins with gastric symptoms. This condition of the stomach and intestines continues throughout the disease.

But this study of stomachic flora seemed to me so difficult that I endeavored to avoid it by making plantings from the organs most attacked, even from those which presented nothing from the anatomic point of view. The first plantings on gelatin of the substance of the liver, kidney, spleen, the mesenteric glands, the walls of the biliary vessels, the blood, and the bile remained sterile, especially in the first cases examined. It was only after continued experiment that I saw appear in the sporadic state, now in one organ, now in another, and always very much disseminated, colonies of a microbe, which I found also when I studied the contents of the stomach and intestines and the famous black vomit which gives to this disease its specific character. I found this microbe with a certain constancy in all cases, and in grave cases it was almost the only inhabitant of the blood contents of the stomach. Moreover, it showed itself pathogenic for the guinea pig.

This fact gave me the idea of isolating it by passage through this animal, and from the first this attempt was attended with good results.

Before describing them, I will allude to an important question suggested to me by Dr. Roux. In the absence of a microorganism in the organs and liquids, is it possible to find a toxic substance circulating in the body which may produce manifestations of the disease? After some fruitless attempts in several directions I drew blood, with a sterilized syringe, from a vein of the arm, prepared as for bleeding, and immediately injected this blood into the peritoneal cavity of a guinea pig. Former experiments showed me that these animals support relatively large quantities of human blood. Ten cubic centimeters of blood taken from a person who was seriously ill, and who died next day, produced in the animal slight malaise, which disappeared next day. A slight rise of temperature of 38.7° to 39.7° continued some days.

In five days the animal had lost 60 grams of its weight, but it subsequently recovered. I repeated this experiment with another patient, also seriously attacked, and with the same success. These facts do not speak in favor of the special efficacy of a toxic substance existing in yellow fever. It then occurred to me that to place a guinea pig, weighing 500 grams, in the same conditions, relatively, to the blood injected, I should inject about 35 grams of the blood of the patient. I repeated my experiments when the patient was dying and injected a guinea pig, weighing 535 grams, with 30 grams of blood. The initial temperature was 38.7°. It rose to 39.9° and remained at this point for two days. On the fourth day the temperature fell to 37.1°. and the animal died. This experiment was repeated with 4 patients severely attacked, the prognosis being doubtful in all the cases. The results in the case of the guinea pig solved the problem, not only as to the existence of a poison, but as to the intensity of the disease. All 4 animals became sick. Two of them are dead, as are also the persons whose blood they received. The two other patients will recover, also the guinea pigs injected. The existence of a toxic substance in yellow fever is, therefore, indubitable.

The most important experiment, with the point of departure for any other researches, is the following:

When we inject under the skin of a guinea pig 1 to 2 c. c. of the contents of the stomach of a person dead of yellow fever, the animal infallibly dies, and we find in his blood, in pure culture, the microorganism which I believe I may consider specific.